

Atypical Myopathy

Unfortunately this week (mid November 2009) has seen the emergence of a disease previously undiagnosed within the practice area. Atypical Myopathy, or Atypical Myoglobinuria as it is also known, is an acute disease that affects the muscles (hence the name - myo-pathy = muscle-disease) of horses and ponies kept at grass, usually occurring in autumn and early winter. Sadly there is a high mortality rate with reports varying between 75% and 90%.

Atypical Myopathy (AM) is still a poorly understood condition. There are reports of it having been recognised as early as the late 1930s with occasional sporadic cases since then. In recent years there has been an increasing incidence of AM in northern Europe and the UK. There is still no proven causative agent although either a fungal toxin or possibly even a toxin produced by the clostridia group of bacteria are suspected to be responsible. Young horse and ponies less than three years seem to be at most risk, especially those around 18 months old, but there have been cases in six and eight year old ponies this week. Affected animals have normally been kept permanently at grass. There may be a link with damp/poorly drained land and possibly with land containing many trees, particularly with a significant amount of dead leaf litter (perhaps due to fungal growth in such conditions). Unvaccinated and poorly wormed ponies may also be at higher risk.

Animals affected show varying signs. Some show signs very similar to colic (Liverpool University have had several cases referred for colic in the last few weeks that have turned out to be AM) whereas others tend to show signs of weakness. This may be as subtle as shifting weight from one foot to another or slight trembling but can be much more severe. One of the cases the practice saw this week was found unable to get up in the field. Darkly discoloured urine is strongly suggested of AM when seen with other clinical signs. This is because when muscle is broken down one of the by-products released is called myoglobin. This is subsequently cleared through the kidneys and released into the urine (myoglobin-uria). Other signs may include a raised respiratory rate and raised heart rate. Strangely, for horses that are showing severe disease, appetite seems to remain quite good with some cases willing to eat while too weak to lift their head. Confirmation of the diagnosis is via blood samples which show large (often massive) elevations in an enzyme called CPK, which is released as a result of muscle breakdown. This is the same enzyme that is elevated in horses that have the condition known variously as "tying up"/ azoturia/ Monday Morning disease. The key difference is that AM sufferers tend to have no history of exercise and the elevations in CPK are generally much more dramatic. Triglyceride, a fat metabolism product, is also often high.

As far as we know death is as a result of respiratory or heart failure when the heart or chest muscles become too weak to maintain their function. Myoglobin can cause severe damage to the kidneys when present in high quantities so kidney failure is another risk, particularly in cases that are not well hydrated.

There is currently no specific treatment to combat the disease. Intravenous fluids help maintain kidney function and assists the horse or pony to pass as much myoglobin out in dilute urine. Plentiful soft bedding is essential as sufferers may spend many hours a

day lying down. Painkillers and anti-inflammatories help keep pain levels down, though it is unclear how painful the condition is. Other treatments such as oral carnitine, injectable riboflavin or metronidazole (an antibiotic that combats clostridia bacteria) have been suggested as possible useful treatments but none have been proven to work.

As far as prevention goes there are several steps that can be taken to reduce the risk, but unfortunately some of them aren't easy or practical to apply. Once a horse or pony has contracted AM that pasture should be considered a high risk pasture and ideally would not be grazed, especially during autumn/winter (and perhaps even spring). If this is not possible then stabling for at least some of the day and feeding concentrates appears to reduce the risk. Supplementing feed with hay should help by reducing the intake of grass, but if the hay is mouldy or fed from the ground during damp conditions it may increase the risk. Permanent stabling is the safest option but has significant cost implications and will often also lead to a very bored and frustrated horse or pony!

We would like to thank Lesley Sanderson from Pendragon Vets in Kirkby Stephen for referring the first case we saw last weekend. Between our practice and Pendragon we have seen three severe cases and one case in a pony that had high CPK levels but was otherwise well. At the moment we are hoping that last week was an isolated outbreak and that there will be no more. While four cases is too small a number to draw meaningful statistics from at the time of writing three out of the four cases have survived, so perhaps there is some ground for optimism.

If you have any questions about AM please get in touch with the practice. As a profession we are still trying to unearth the secrets of what causes AM and how it affects the horse. There are inevitably still lots of unanswered questions but we will do our best to pass on what we currently know to enable you to keep your horse or pony as safe and well as possible. We will try to keep this site updated if or when we hear of more cases.

There is a Belgian website that gives more information on the disease - <http://www.myopathieatypique.be/fr/souscategorie.php?categorieID=15>